modified Hodge test was 0.016–32 mg/L. The positive isolate was the only isolate with a meropenem Etest MIC >2 mg/L.

Four isolates were referred to ARMRL for further characterization (Table 1). Two isolates were confirmed as carbapenemase producers and two were not confirmed.

For the majority of isolates for which a confirmatory meropenem Etest was performed, the Etest MIC was lower than the Vitek 2 MIC, suggesting that Vitek 2 over-calls meropenem MICs. Most Enterobacteriaceae isolates with a raised meropenem MIC by Vitek 2 in our laboratory were not confirmed to have true carbapenem resistance on further investigation. We concluded that it would not be justified to perform the full range of confirmatory tests on all such isolates, but that we should perform an Etest and refer for further testing only if the Etest MIC was ≥1 mg/L, one dilution higher than the guidelines.1,2

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No specific funding was received for this study. All data were generated as part of our routine work.

Transparency declarations
None to declare.

References

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No NDM-1 carriage in healthy persons from Mumbai: reassuring for now
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Keywords: ESBLs, carbapenems, ertapenem

Sir,
We read with interest the article on the faecal carriage of NDM-1 Enterobacteriaceae from patients in a military hospital in Pakistan, implying the potential for spread in the community. At a tertiary care centre in Mumbai, we screened for the presence of carbapenem resistance over a 6 month period (January to June 2011) by examining 1000 consecutive faecal samples from healthy individuals (not patients) reporting for routine health screening. This study was ethically approved by the National Health and Education Society, P. D. Hinduja National Hospital and Medical Research Centre.

Stool samples were cultured on MacConkey agar and incubated at 37°C for 24 h. Enterobacteriaceae were then tested for the presence of extended-spectrum β-lactamases (ESBLs) on Mueller–Hinton agar using cefpodoxime discs and were further evaluated using β-lactam inhibitors, i.e. ceftazidime and ceftazidime plus clavulanic acid, and cefotaxime and cefotaxime plus clavulanic acid. According to the CLSI 2011 guidelines (M100-S21), disc diffusion testing was undertaken with a 10 µg ertapenem disc placed on a bacterial lawn prepared using a bacterial suspension with a turbidity equivalent to that of a 0.5 McFarland standard (1.5×108 cfu/mL). The zones around the disc were assessed after incubation at 37°C for 24 h. If isolates were identified as carbapenem resistant, they were to be molecularly characterized for the presence of the blaNDM-1 gene.1

Of the total number of samples, 23.9% (239/1000) were found to be ESBL producers, of which 95% (227/239) and 5% (12/239) were identified as Escherichia coli and Klebsiella species, respectively. None of the 1000 isolates was carbapenem resistant. The findings of our study are contrary to those reported by Perry et al.,1 where a high prevalence (18.5%) of faecal carriage of carbapenem-resistant Enterobacteriaceae was noted, albeit among patients. However, an earlier report from our institute reported the absence of NDM-1 in follow-up stool samples of 10 patients who had earlier been diagnosed and treated for infections caused by NDM-1-producing Enterobacteriaceae.2

A study done at our institution in 2004 demonstrated an ESBL faecal carriage rate of 11% in a similar cohort of individuals reporting for routine health screening.3 This carriage rate has increased to 23.9% in antibiotic-naïve healthy persons. Fortunately, at least in our small study, NDM-1 has not spread to the same extent. Though this is reassuring, it should not detract from the need to implement strict infection control measures and antimicrobial stewardship for resistant organisms.

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Transparency declarations
None to declare.
Lack of effect of extracorporeal membrane oxygenation on tigecycline pharmacokinetics

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Keywords: antibiotics, respiratory tract infections, RTIs

Sir,

Extremely limited data are available on the pharmacokinetics of antimicrobial agents during extracorporeal membrane oxygenation (ECMO). We report the case of a young adult patient who was admitted on day 1 to our intensive care unit for acute respiratory failure due to intra-alveolar haemorrhage. Ventilator-acquired pneumonia due to Staphylococcus epidermidis and Staphylococcus warneri developed, initially treated with intravenous vancomycin. Despite maximal ventilator settings, optimal respiratory function could not be obtained, so veno-venous ECMO was started on day 38. The system comprised a membrane oxygenator (Quadrox Bioline, Jostra-Maquet, Orleans, France) and a centrifugal pump (Biomedicus 560, Medtronic, Minneapolis, MN, USA). Yet on day 50, respiratory conditions worsened. A persistent S. epidermidis pulmonary infection was observed, with induction of vancomycin resistance requiring antibiotic change to tigecycline on day 55. The patient’s renal function remained stable during hospital stay (ClCR 95 mL/min), but despite therapy and complete bacteria eradication the patient’s condition continued to deteriorate until death occurred on day 61. Tigecycline concentrations were measured by liquid chromatography–tandem mass spectrometry in plasma and tracheal aspirate.1 The Berkeley Madonna software (v 8.3.18, University of California, Berkeley, CA, USA) was used to predict tigecycline concentrations using population mean parameter estimates from a previously published pharmacokinetic study in critical care patients.2 The tigecycline dosing regimen and concentrations are presented in Table 1. Interestingly, concomitant tigecycline concentrations measured in plasma and aspirate were virtually identical.

The potential effect of ECMO on tigecycline pharmacokinetics had never been investigated before. Tigecycline is characterized by a large volume of distribution with a population mean value of 398 L in critical care patients,2 which is therefore unlikely to be noticeably increased simply by dilution into the system, but adsorption on the circuit membranes could not be excluded.3,4 However, measured tigecycline plasma concentrations were virtually similar to the values predicted for a critically ill patient with ClCR=95 mL/min and body surface area=1.42 m2,5 suggesting that ECMO has no effect on tigecycline pharmacokinetics.

Table 1. Plasma and tracheal aspirate concentrations of tigecycline administered intravenously at a dose of 50 mg twice daily simultaneously to ECMO

<table>
<thead>
<tr>
<th>Measured tigecycline concentrations</th>
<th>Predicted tigecycline concentrations from Rubino et al.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 58, time since treatment initiation</td>
<td>Plasma concentrations, mg/L (time since last dose) 0.50 (2.5 h) 0.26 (8 h) 0.21 (11 h)</td>
</tr>
<tr>
<td></td>
<td>Plasma concentrations (mg/L, time since last dose) 0.31 0.24 0.22</td>
</tr>
<tr>
<td>Day 56, time since treatment initiation</td>
<td>Plasma concentrations, mg/L (time since last dose) 0.20 (11 h)</td>
</tr>
<tr>
<td></td>
<td>Plasma concentrations, mg/L (time since last dose) 0.31 (4.5 h) 0.37 (5 h) 0.26 (8 h)</td>
</tr>
<tr>
<td></td>
<td>Aspirate concentrations, mg/L (time since last dose) 0.23 0.28 0.25</td>
</tr>
</tbody>
</table>

References


